Key words : Transient global amnesia (TGA), Early repolarization syndrome (ERS), Cardiac autonomic nerve activity (CANA)

The transient global amnesia and the early repolarization syndrome ; A consideration on causes of transient global amnesia both from several cases that author experienced and references

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Introduction

There is a syndrome called transient global amnesia (TGA). This syndrome shows transient anterograde amnesia without organic brain disorder, but it completely recovers to previous health condition without recurrence. Though many hypotheses were reported on a cause of TGA, a hypothesis of the transient ischemia of hippocampal cingulate gyrus is well known above all. Furthermore, TGA is said to occur mainly in middle age or old person, very rare in young generation. However, for the moment, the true cause of TGA is still unknown. On the other hand, a unique syndrome that is called early repolarization syndrome is well known in an electrocardiogram characteristic from old days. Early repolarization syndrome (ERS) is defined from J waves and ST elevation in two or more in a posterior and lateral leads of electrocardiogram. Previously, this type of ECG has been thought to be a normal variant. When this pattern is seen in V_4-V_6, it is treated as a relatively safety variation of ECG. However, it is recently known ERS induces sudden death by ventricular fibrillation (VF), and relation to Brugada syndrome is being discussed. First, the author describes an interesting case of a TGA that has ERS as the complication in a young woman, which experienced recently. Next, the author talks about various medical hypotheses of TGA from references, and showed this case meets the criteria as TGA. In addition, the author presented the other several cases that thought to be TGA, which experienced recently. Last, the author attempted the comparison of those cases with the first case.

The case report

Sixteen years old female, a high school girl, she does not have particular previous history, and the family history.

Clinical history : One day she joined the marathon competition of her high school. She started as usual, but she began to feel an absent-minded

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feeling at the third round of the track. She reached a goal point after all. However, she did not remember completely her own behavior. After the competition end, she asked a friend "Why I am here and what I was doing here" repeatedly. She had an experience of similar behavior during jump rope in the middle-school age. Furthermore, one day, she experienced the numbness of hands and feet for about one hour. Moreover, one time, a friend told that your breathing is somewhat strange. She often felt the shortness of breath, and tachycardia around 100 bpm when walking. She visited the author's clinic with wish to remove these various discomfort manifestations.

Present illness : Her physique was moderate ; 164 cm in height, 59. 9 kg in weight and the body mass index (BMI) was 22. 3 kg/m^2, and her consciousness was clear. The eyelids and bulbar conjunctiva were not either anemic or icteric. The oral cavity was normal, cervical lymph nodes were not swelling, jugular vein was not dilating, and goiter was not touched. The blood pressure was low in 90-60 mmHg, but the value was not different in both arms. The heart size was normal, and CTR was 48. 4%. The heart rate was 78 bpm, regular rhythm, and heart sound pure, but the first sound was splitting. A heart murmur and an extra-heart sound were not audible. Breathing sound in lung was normal, and rale or noise was not audible. Hepatosplenomegaly and percussion pain was not detected. Pretibial edema and abnormal neurological sign were not observed.

Laboratory examination : Results of laboratory examinations are as follows ; RBC 412×10^4/μl, Hb 12. 9 g/dl, Ht 39. 1%, MCV 95, MCH 31. 3, MCHC 33. 0, WBC 5, 970/μl (Baso 0. 5%, Eos.2. 8%, Lym. 40. 4%, Mono 7. 7%, Neutro 48. 6%) AST 23 U/L, ALT 17 U / L, LD 198 U / L, CK 201 U / L, TC 208 mg / dl, HDL-c 78 mg / dl, LDL-c 114 mg / dl, L / H 1. 46 , TG 47 mg / dl, CRP ＜ 0. 05 mg / dl, NT-proBNP 49 pg/ml.

Clinical analysis of this case

Table 1 shows the change of heart rate, blood pressure, and cardiac autonomic nerve activity (CANA) in an orthostatic endurance test. In the orthostatic endurance test, one minute after posture change, CVRR is decreased, the RR interval is shorted, and the heart rate is increased ; that suggests sympathetic nerve tension occurs as a reflex. On the other hand, systolic blood pressure is fall in 21 mmHg, and it becomes less than 90 mmHg (80 mmHg). This fact suggests she has an orthostatic dysregulation as a background.

Table 1 The orthostatic endurance test ; one minute after posture conversion from the recumbent position to the orthostatic position ; blood pressure decreased, but heart rate increased.

	CV-RR	SDNN (SDRR)	Mean RR	Max. RR	Min. RR	Mean. HR	BP. (mmHg)
Recumbent	5.21%	48 ms.	980 ms.	1036 ms.	820 ms.	63 bpm.	109/69
Orthostatic	4.59%	320 ms.	707 ms.	780 ms.	660 ms.	84 bpm.	88/65

Fig. 1 shows an ECG that recorded at a rest state. The ECG shows ST elevation with an asymmetric QRS wave (in **Fig. 1-a**), and ST segment in aVR is depressed conversely (in **Fig. 1-b**). In V_4, ST shows concave elevation accompanied by a notch (J) of the early ST segment (in **Fig. 1-c**). These findings are characteristic on ERS.

Then, the author performed Master's double 2-step exercise test for her. She finished the test without specific complaints, and the heart rate only reached 64% of the target. Therefore, her tolerability to an exercise was thought to be not so poor. Second, the author attempted to induce the same symptom through the same running exercise by using a Holter ECG. With an agreement of herself, the author asked her to attach

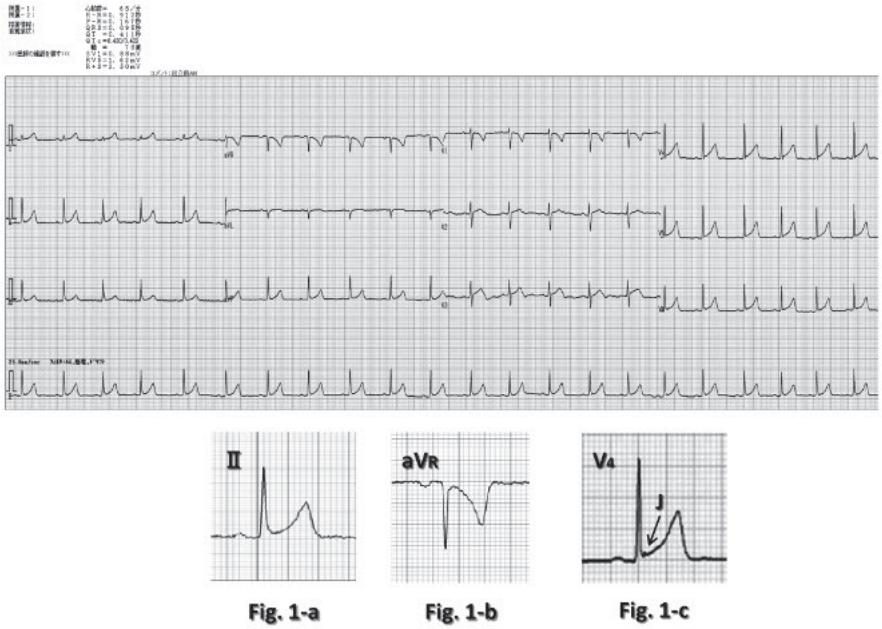

Fig. 1 ECG at initial diagnosis; Fig. 1a-1c shows characteristics of an early repolarization syndrome (ERS).

Holter ECG equipment, and to try a marathon of 3.5 km in a truck of her high school. She started the training at 11:00 and the heart rate increased gradually; soon after, it began the sudden increase and showed the minimum RR interval in 220 msec (273 bpm as heart rate) at 11:52. Though she felt strong palpitation and shortness of breath, training was completed without a disturbance of consciousness. Her memory was kept during the exercise.

Fig. 2 demonstrates the record of Holter ECG including tachycardia. Recorded tachycardia in Holter ECG is narrow QRS preceded with P waves. Therefore, that is thought to be atrial tachycardia or inappropriate sinus rhythm.

Fig. 3 shows a change of the heart rate. Start at the test is 11:00, the onset of the tachycardia is the latter half of 11:18, and it finished at around 11:55. The author recorded whole heart rates including recovery time through the test. The recorded time is 90 minutes, total heartbeat is 12,511, and the average heart rate is 149±35 bpm. The maximal heart rate 273 bpm was recorded at 11:52:32 in the 7,621th beat (conversion value as the RR interval is 220 msec).

Fig. 4 is a part of Holter ECG that was recorded during the night. The J wave is remarkable in lead of CM5. Furthermore, this Holter ECG shows the repeat of first-degree AV-block and normal conduction.

Last, as mentioned in Fig. 3, the author converted whole heart rate to the RR interval, within exercise including tachycardia. Then the author divided this RR data to each one-minute unit, and calculated the cardiac sympathetic nerve activity (CSI), the cardiac vagus nerve activity (CVI), and an integrated cardiac autonomic nerve activity (CANA) by using each unit from the Lorenz plot method[1)~3)]. In addition, CANA means cardiac autonomic balance that calculated as CSI/CVI (ratio). Therefore, when CANA is high, the sympathetic nerve activity is strong, and when CANA is low, the vagal nerve activity is strong.

Fig. 5-a shows the relationship between heart rate change and CSI. The left longitudinal axis shows heart rate, and right axis shows CSI. Be-

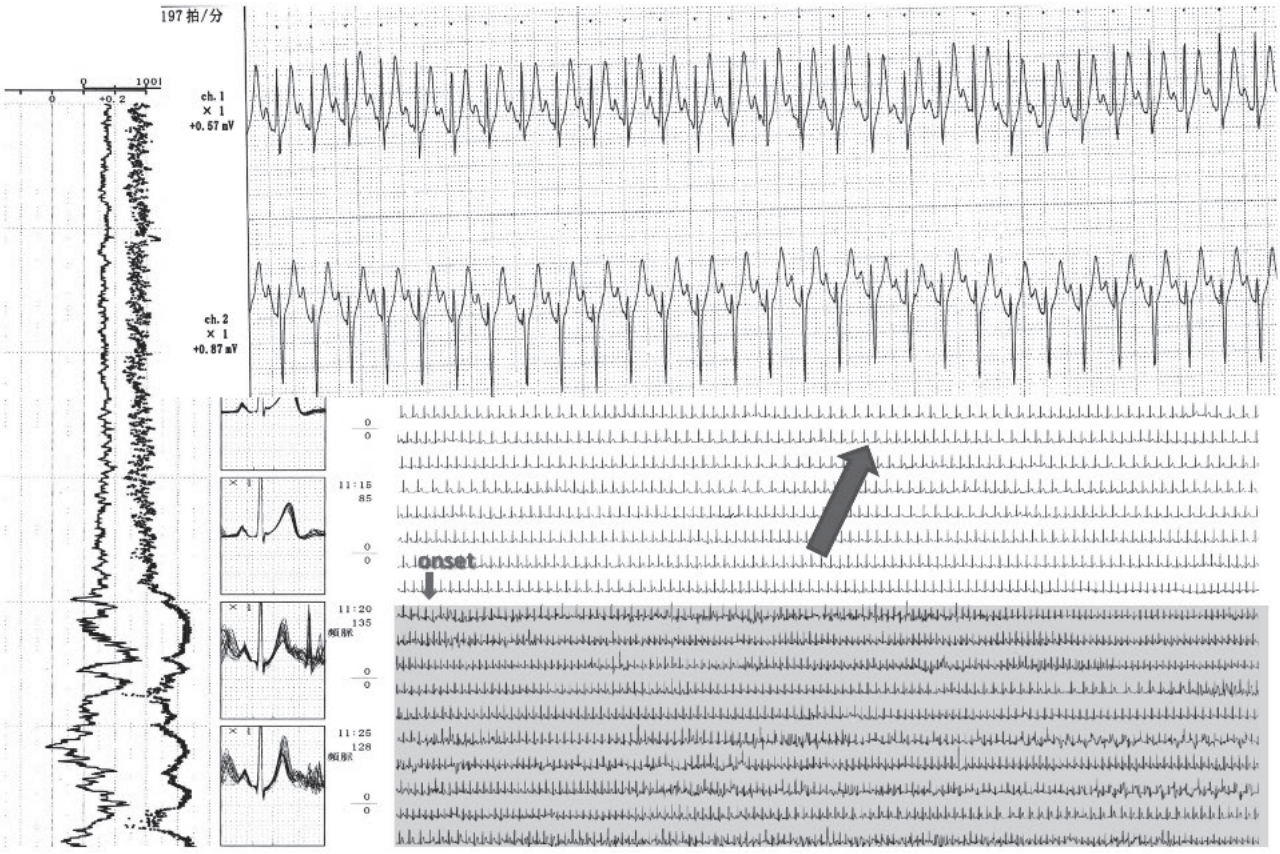

Fig. 2 Holter ECG shows the onset of the tachycardia attack and the enlarged picture of it; the tachycardia is narrow QRS, and it accompanies p preceding the QRS wave. It means this tachycardia is an atrial origin.

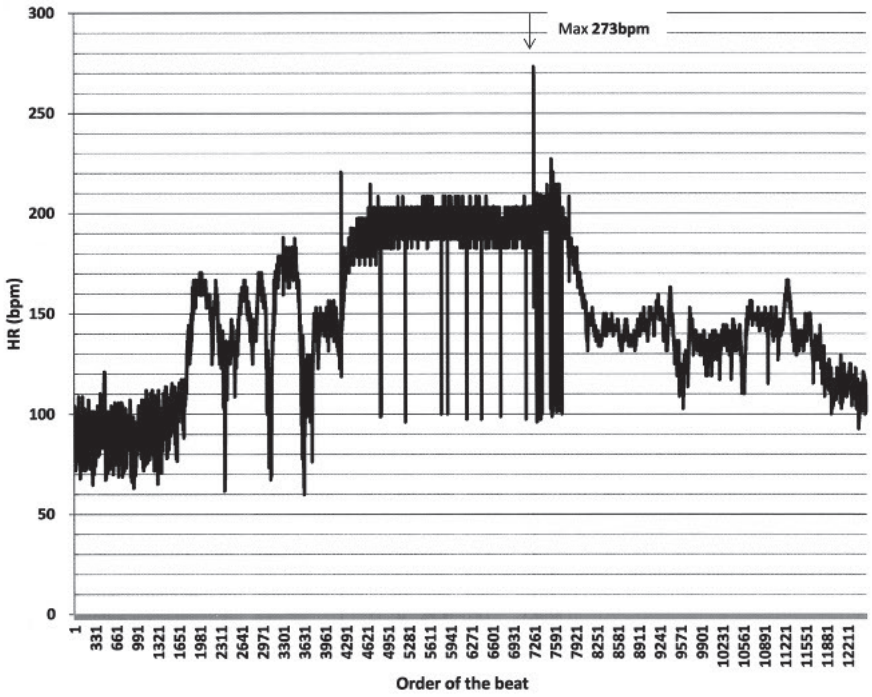

Fig. 3 Figure shows heartbeat change including the tachycardia; the total numbers of heartbeats are 12,511 per 90 minutes, and maximal heartbeat reaches 273 beat per minute (arrow).

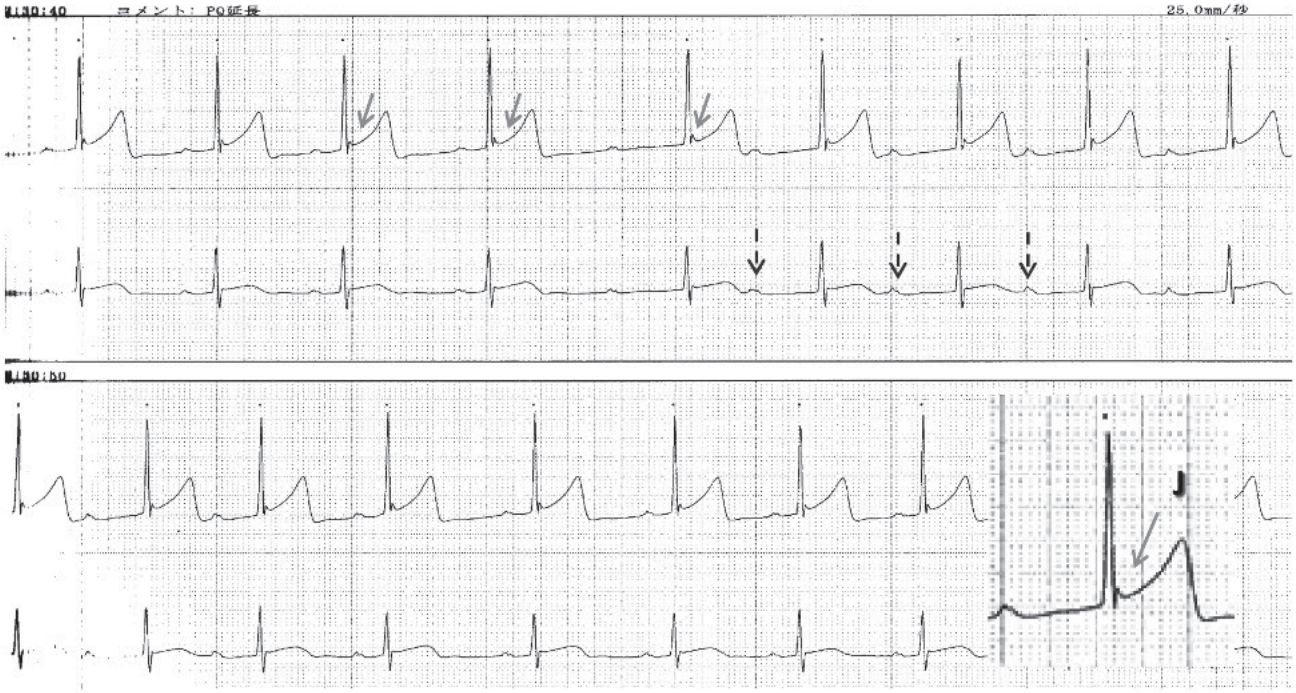

Fig. 4 A part of Holter ECG in night; ECG shows prolonged PR intervals (First-degree AV block). A dashed line arrow shows the P wave that precedes the QRS interval, and a simple arrow shows the J wave that becomes more obvious in vagotonic condition.

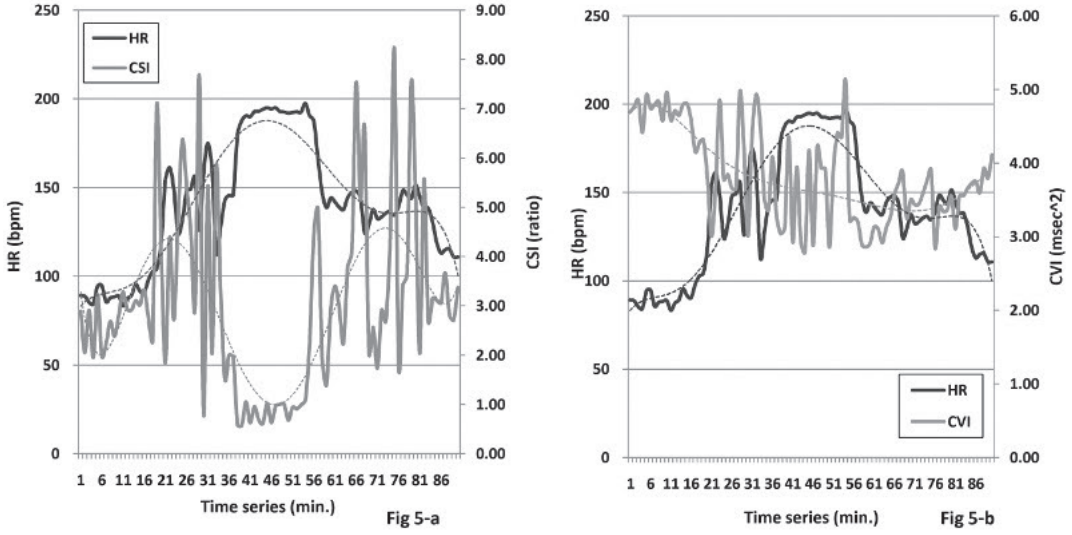

Fig. 5-a The heart rate and the CSI change along the time axis; each of them shows the mirror image during the tachycardia. CSI shows a form of a remarkable vibration wave before and after the tachycardia.

Fig. 5-b The heart rate and the CVI change along the time axis; CVI shows a remarkable vibration during the tachycardia inversely to CSI, and that vibration is small before and after the tachycardia.

fore the onset of tachycardia, the sympathetic nerve activity fluctuates intensely, and shows a state like a storm. Subsequently, CSI decreases remarkably, and shows low activity without almost fluctuation during the tachycardia attack. Before the end of the tachycardia, intense storm in CSI occurs again, and returns to physiological fluctuation finally.

Fig. 5-b shows the relationship between heart rate change and the vagus nerve activity (CVI). The axis definitions are equal to Fig. 5-a. in both the longitudinal main axis and the second axis. CVI before and after the onset of the tachycardia is stable physiological fluctuation in contrast to CSI, but it falls in the intense autonomic nerve storm during the tachycardia attack.

Fig. 6 shows the relationship between heart rate change and CANA. In the rest condition of healthy people, from the experience and gathered data analysis until now, the author thinks vagus nerve activity is approximately double compared with the sympathetic nerve activity. Therefore, the standard value of CANA is thought around 0.5. During normal heart rhythm, CANA shows stable fluctuation physiologically, but the onset of tachycardia induces the remarkable change of CANA. When the onset of the tachycardia, CANA moves to the dominant direction in vagus of small fluctuation, and shows a mirror image against the heart rate curve. On the recovery time after exercise, CANA returns to the previous stable condition after the relatively long interval with remarkable fluctuation.

Fig. 7 shows correlation between the heart rate and CANA from the multinomial regression analysis. A quadratic equation is most suitable to explain this correlation; see the equation on the graph of Fig. 7. If rising of the heart rate is under around 140 bpm, CANA stays sympathetic nerve dominant side. When the heart rate is over about 140 bpm, CANA moves to the vagus dominant side. Namely, these findings suggest sympathetic nerve changes unto parasympathetic nerve (vagus).

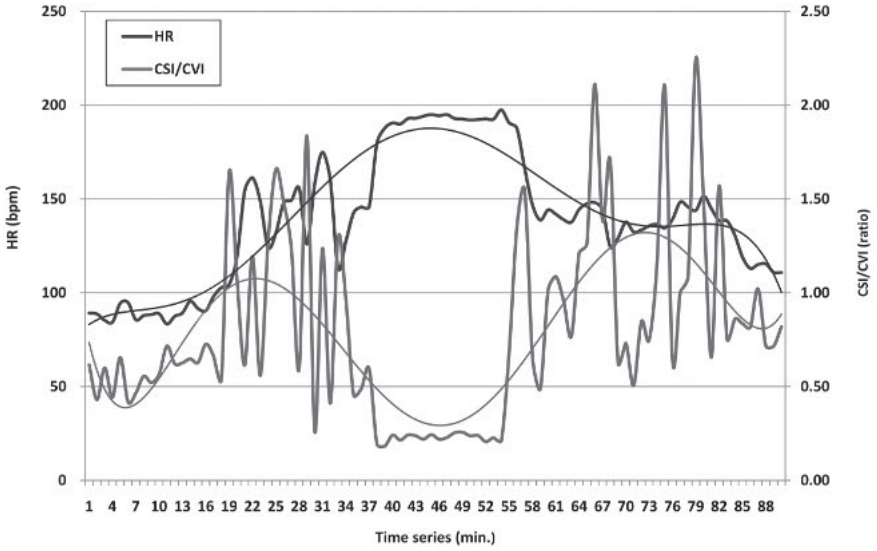

Fig. 6 The changes of CANA (CSI/CVI) on the time line; the heart rate and CANA show the remarkable mirror image. CANA moves to the vagal side during the tachycardia; it is thought to be a reaction to suppress the excess heart rate. On the other hand, CANA shows remarkable fluctuation (autonomic nerve storm) before and after tachycardia.

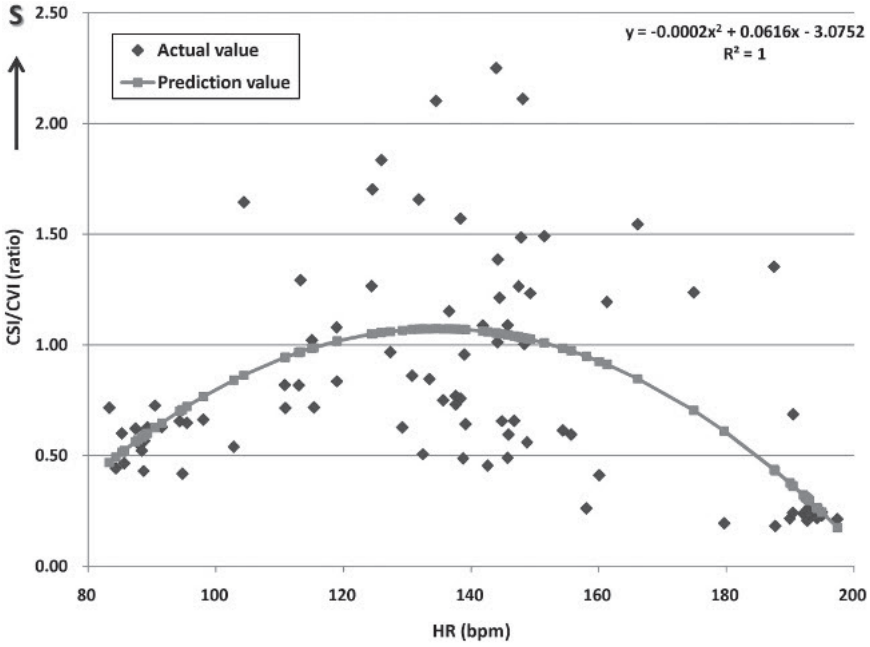

Fig. 7 The regression equation of CSI/CVI from the scatter diagram of heart rate; increase in sympathetic nerve tension induces the increment of heart rate. However, the autonomic nerve activity tends to the vagal side from 140 bpm in heart rate.

The integrated discussion of causes of TGA including the author's experience

Transient global amnesia (TGA) is an interesting syndrome. The main manifestations of this syndrome are as follows; obvious organic brain disorder is not seen, a patient shows transient anterograde amnesia, and the patient develops the disability of circumstantial judgment, and forgets the behavior that oneself had done. Therefore, the patient asks questions repeatedly; "Why am I here?" "What time is it?" "When I came here?" alternatively "How did I come here?" The answers are immediately forgotten by the patient's memory disability. Despite such a memory disability, patient keeps physical behavior in normal during the attack. Bender BM[4] reported the first case of this disorder in 1960, and Fischer CM and Adams RD named it TGA, in 1964. Many a researcher is reporting TGA is onset mostly in 50s' female, and it recovers almost in several hours without sequelae, despite the complete lack of memories during the attack, and recurrence is rare. In the author's case, clinical manifestations are as follows; there is an eyewitness of the attack, and transient anterograde amnesia is obvious, higher brain function disorders except amnesia are not seen, previous histories of epilepsy or brain trauma are not seen, and the attack recovers without sequelae soon. This characteristic is almost equal in the diagnostic criteria of TGA except her age, so the author diagnosed this case as TGA despite young. The cause of TGA is still unknown, but several researchers are reporting that the memory related part of the temporal lobe and hippocampal cingulate gyrus shows ischemia during the TGA attack[5]. Though the origin of ischemia of the hippocampal area during the TGA attack is still unclear, the patient often accompanies a migraine. Therefore, vasoconstriction in migraine induces hypo-perfusion in the temporal lobe, and it causes a reduction of brain oxygen

metabolic rate, and tissue oxygen extraction[6]. These clinical conditions are common as manifestations in TGA. Though it is not whole case, patients of TGA in the early phase of the attack, often show the high signal in the hippocampal area in MRI. The signal is detected in a diffusion-weighted image (DWI) and T2 weighted image (T 2) and FLAIR image. However, these findings are reversible, and are not persistent specifically in T2 or FLAIR. These findings in TGA are differing from the transient ischemic attack (TIA). Therefore, it suggests that TGA is not occurring from ischemia, and not worsening from ischemia to an infarction[7]. Toledo M and coworkers examined 28 cases of TGA patients by means of carotid and intracranial Doppler echo, DWI, a perfusion-weighted image (PWI), and angiographic MRI. They reported that any perfusion abnormalities, stenotic lesion in the artery, or the cardiogenic cerebral embolism are not seen, as the results. Therefore, they concluded the arterial lesion is unrelated to TGA[8]. On the other hand, a hypothesis of the venous stasis as the cause of TGA is always influential. This hypothesis says elevating intrapleural pressure by such as a Valsalva maneuver intercepts blood return in superior vena cava. In addition, the insufficiency of the cervical venous valve induces the reverse flow of the vein. Subsequently, saying that pressure of the inside temporal vein rises and disturbs the hippocampal function as the results[9]. However, despite many hypotheses as mentioned above, the true cause of TGA is still unclear. Therefore, observations of clinical manifestations through attack are most important to diagnose TGA. Some emotional stimulation (such as sexual intercourse), physical stimulation (an extreme exercise), or perceptual stimulation (such as pain) is said to induce TGA. Furthermore, abnormal head posture in long time or a sort of medical intervention are said to induce TGA[10]. For example, Chan-Ying C and coworkers are reporting the onset of TGA after catheter ablation that had performed as a treatment for paroxysmal supraventricular tachycardia[11]. Sealove[12] and coworkers reported a case that repeats TGA after an operation of the heart. This patient was showing a remarkable hyper-fibrinogenemia and the increase of blood sedimentation rate. Moreover, the patient showed the TGA attack in standing position, but recovered in the recumbent position soon. Furthermore, the TGA attack ended with recovery of these abnormal laboratory findings. From above findings, they insist that abnormal blood circulation in the brain induces TGA. From the general report, the incidence of TGA is 3.4-10.4 people per 100,000 people per year. The onset is almost in middle age or the elderly, but very rare in young. From a research study that performed on Japanese, Ohsato A. and coworkers reported 175 cases of TGA. In their study, the most is 90 cases of TGA in 60s. However, the case of TGA is not detecting from less than 30 years old[13]. On the other hand, there are several reports that TGA occurs even if a young person, specifically if they are sports player. For example, a case of 21 years old college baseball player (he is suffering type 1-diabetes), 16 years old boy football player, and 13 years old girl volleyball player was reported by each researcher[14),15]. The onset of TGA in these sports players occurs during each competition, and accompanied migraine as shared characteristics. In the author's case, the patient is young, and the onset of TGA occurred in exercise. These findings are matching with above-mentioned cases, but the author's case did not have epilepsy or migraine in previous history. However, she has experienced hyperventilation like symptom, a numbness of limbs, and absent-minded feeling previously. These symptoms probably suggest anxiousness and tension as her

mind background. Therefore, it is probable of an omen of a migraine despite atypical. Whether this case is TGA or not, the author thinks she has anxiety and the tendency to autonomic disorder. Therefore, the author attempted to estimate her psychosomatic condition. First, the author performed the easy psychological test. She showed a trend of psychosomatic disease in the Cornell Medical Index (CMS; a health survey test). Moreover, she showed a depressive tendency from the self-rating depression scale (SDS). Next, in an orthostatic endurance test, despite decrease in her heart rate fluctuation, heartbeat itself increased. Furthermore, secondary hypotension occurred, and these findings filled the criteria of an orthostatic dysregulation (O. D). Therefore, she has above-mentioned psychosomatic unstable characteristics. In addition, ST elevation with a J wave and asymmetric ORS are seen in the chest leads, so the author thought this case is an early repolarization syndrome. Unfortunately, the author failed to induce TGA though the heart rate increased remarkably by exercise. In the author's case, the true origin of TGA is unclear, but it is probable that J wave syndrome induced causative arrhythmia (maybe tachyarrhythmia) and caused TGA due to local hypoperfusion in the hippocampus area. Generally, if atrial tachycardia as above or faster atrial rhythm occurred, ventricular reply to the atrial stimulation is difficult. On the other hand, an extreme exercise increases muscle oxygen demand, and blood distribution becomes more abundant in the motor system than the other organs. Moreover, shortened diastolic interval by tachycardia decreases blood return to the heart. Cardiac output reduces as the result, and it induces local ischemia of the hippocampus area and causes a TGA attack. Therefore, the theory of the local hypoperfusion is one of the mechanisms that the author thought about this case. In addition, the background factor of this case is including complicated findings such as a depressive neurotic character, anxiety from the previous experience, disorders of the heart rhythm due to J wave syndrome, orthostatic dysregulation, and an autonomic nerve storm. These characteristics probably contribute also the onset of TGA. This time, the author could not perform further examination such as a brain imaging. However, generally, a diagnosis of TGA is being based on clinical findings, and this case is filling clinical characteristics. Even if survey instruments found some abnormal findings, its duration is short, and the change is almost within as the early phase of onset. Therefore, the author concluded this case is a TGA. Previously, ERS was thought as a normal variant. Therefore, this syndrome was not attracting general attention. However, in recent years, ERS attracts attention with relationship to Brugada syndrome that often induces sudden death from fatal arrhythmia[16]. Some researchers (Juhás S. and coworkers) reported that 7% of the neurosis patient has J wave syndrome. Those patients increased heartbeats due to the orthostatic dysregulation, and showed hypotension remarkably during the orthostatic endurance test. Moreover, they say that 50% of the neurosis patient shows sinus tachycardia and 30% shows supraventricular extra-systole, and say the systolic function on the echocardiographic parameters also increases.

Recently, the author experienced three cases of TGA except in above case. The first case occurred in a young male of the American football player. This case also had J wave syndrome. One day, he suddenly fell down during the practice game. His consciousness looked normal, but he had hit the head. So, he was examined from the neurological diagnosis, and brain imaging. Despite the result of examination normal, he asked his friend and trainer repeatedly "Why am I wearing the uniform like this" or "Why am I in

this hospital now". The neurologist explained to his trainer that this symptom is probably transient. Next day, he recovered, but he was completely being lost the memory of incident of the yesterday. Previously, on his high-school days, he was involved to the sudden tachycardia attack during the soccer game, and the author had an experience that diagnosed him as ERS at the time.

The second case is 60's male. Usually, the author is treating him for hypertension. One day when he was doing "Mahjong" with friends, friends noticed his behavior is strange in something. He asked those friend questions repeatedly as "Why am I here?" and "Why am I doing mahjong". However, he continued the game with no problem and ended as usual, but he completely forgot a way to return for his own home. That is why, a friend led his motorbike and sent to his home. He could operate the motorbike completely as usual, and could follow to the friend. Next day, the wife asked him; "Do you remember what behavior you did yesterday?" However, he could not recall it at all. Therefore, the author consulted a neurologist about his symptom. The neurologist examined him, but any neurological disorder, the abnormal sign, or the organic brain diseases were not found. The doctor diagnosed him as TGA after all.

The third case is 80's female. Her basic illness is valvular heart disease accompanied by chronic atrial fibrillation. One day, she visits the author's clinic as usual, and left for her home after practice. By chance, her neighbor found her at the station of the suburban train. That time, she could not understand how to buy a train ticket, was standing with a perplexity state. The neighbor bought a ticket for her to take back her to the home. Subsequently, she was speaking with the neighbor as usual on the suburban train. Next day, her family asked on her strange behavior of yesterday, but she could not recall it. The author thought this symptom is TGA. However, she is being persistent atrial fibrillation, so the author thought this episode needs to do differential diagnosis from TIA including a small cerebral embolism. The author consulted a neurologist, and he said that both the neurological sign and abnormal images in brain were not found in this patient. He said this case is probably TGA from the viewpoint of symptomatology, but said that it needs rule out diagnosis from temporal lobe epilepsy. Therefore, the author is continuing observation now.

Additional remarks as a conclusion

From the author's experience and researcher's hypothesis, the author thinks that TGA is not so rare illness, and it occurs even if young people. In young people, probably extreme exercise induces TGA, and often accompanies abnormal ECG such as ERS. Moreover, we should not ignore psychological elements or autonomic nerve disorder as a cause of TGA. Furthermore, we should not forget a migraine is a principal complication of TGA in young people. On the other hand, aged people often have an organic brain disease or brain circulation disorder as a background of TGA. Therefore, TGA needs differential diagnosis from TIA at emergency practice. The author thinks hypoperfusion of the hippocampal area is one of the important causes of TGA whether young people or the aged person as the conclusion.

Acknowledgement

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COI: The author is not relation in conflict of interest in this report.

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一過性全健忘と早期再分極症候群；一過性全健忘の原因について，著者の経験した数例と文献からの考察

金子 仁*

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16歳女性，主訴：一過性前向性健忘．学校の耐寒競技でトラックを走っていてボーッとした感じになった．友人の話ではそのまま走行を続け，競技そのものは無事終了したがその間の記憶は全くない．意識回復後は全く後遺症的なものはなかった．以前にも運動中に同じような事が度々あったので心配になって受診．安静時心電図で早期再分極症候群（J波症候群）を認めた．ホルター心電図を装着し，同様な運動をさせて再現試験を行った．意識障害は起こらなかったが最大心拍数273 bpmに達し，先行P波を伴う上室性の頻脈を認めた．頻脈発作中およびその前後90分間に渉り記録した全心拍（12, 511 beats）のRRから交感・迷走に分けて心臓自律神経活性変動を分析した所，激しい自律神経ストームを認め，さらに交感神経と迷走神経では変動様相が全く異なった．本症例は一過性全健忘（Transient Global amnesia；TGA）と考えられた．今回この発作の原因について検討した結果に加え，著者の経験した同じく一過性全健忘と思われる数例について述べ，文献的考察を交えてその原因について検討した．また，従来若年者には稀とされていた一過性全健忘が若年者にも起こり得ること，若年者においては早期再分極症候群のような心電図異常を有し，しばしば運動に誘発され，偏頭痛を伴う事，さらには不安や鬱傾向のような精神的要因，自律神経失調などが発症の誘因若しくは背景となる事について述べた．

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